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10-Year prospective study of noise exposure and hearing damage among construction workers

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Abstract

Objectives—To characterise the effects of noise exposure, including intermittent and peaky exposure, on hearing damage as assessed by standard pure-tone thresholds and otoacoustic emissions, a longitudinal study was conducted on newly hired construction apprentices and controls over a 10-year period.

Methods—Among the 456 subjects recruited at baseline, 316 had at least two (mean 4.6) examinations and were included in this analysis. Annual examinations included hearing threshold levels (HTLs) for air conducted pure tones and distortion product otoacoustic emission (DPOAE) amplitudes. Task-based occupational noise exposure levels and recreational exposures were estimated. Linear mixed models were fit for HTLs and DPOAEs at 3, 4 and 6 kHz in relation to time since baseline and average noise level since baseline, while controlling for hearing level at baseline and other risk factors.

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Results—Estimated L_{EQ} noise exposures were 87 ± 3.6 dBA among the construction workers. Linear mixed modelling demonstrated significant exposure-related elevations in HTL of about 2–3 dB over a projected 10-year period at 3, 4 or 6 kHz for a 10 dB increase in exposure. The DPOAE models (using $L1=40$) predicted about 1 dB decrease in emission amplitude over 10 years for a 10 dB increase in exposure.

Conclusions—The study provides evidence of noise-induced damage at an average exposure level around the 85 dBA level. The predicted change in HTLs was somewhat higher than would be predicted by standard hearing loss models, after accounting for hearing loss at baseline. Limited evidence for an enhanced effect of high peak component noise was observed, and DPOAEs, although similarly affected, showed no advantage over standard hearing threshold evaluation in detecting effects of noise on the ear and hearing.

INTRODUCTION

Despite a widespread recognition of the impacts of noise on hearing and other aspects of health, noise exposure in industry remains a significant problem,¹² especially in the construction industry³ where noise exposure levels of about 87 ± 5.5 dBA L_{EQ} have been documented.⁴⁵ Noise-induced hearing loss (NIHL) among construction workers is common, with rates from 30% in operating engineers⁶ to over 50% in labourers.⁷ Larger, multitrade US studies have demonstrated prevalence of over 50%,⁸ and studies in countries such as Taiwan⁹ and Germany¹⁰ have shown similar prevalence of between 40% and 60%.

Otoacoustic emissions have held promise as an objective measure of early, and perhaps, preclinical cochlear compromise since the late 1970s.¹¹ In animal and human studies, reduced emission levels have been observed following damage to the outer hair cells in relation to exposure to noise or other ototoxic agents, even in the absence of changes in hearing threshold levels (HTLs).¹²¹³ There is continuing interest in otoacoustic emissions as an early indicator of damage and as a method of screening individuals for susceptibility to NIHLs.^{14–16}

Much of the literature on NIHL is derived from cross-sectional studies, providing limited evidence for the natural history of the condition, a paucity of contemporaneous noise exposure measurements, and a lack temporal specificity. Longitudinal cohort studies are preferred as a method for understanding the development of an environmentally induced condition, especially those such as NIHL, which may follow a non-linear natural history.^{17–19} Models suggest that hearing loss, particularly in the critically noise-sensitive frequencies (ie, 3–6 kHz), rises rapidly over the first 10 years of exposure, slowly levelling towards an asymptotic level of damage, depending on the noise intensity.²⁰ Additionally, peak sound pressure levels may produce greater damage in excess of that predicted by the L_{EQ} .²¹²²; however, national and international bodies support the idea that all noise contributes equally to damage, as reflected in the 3 dB exchange rate used in the L_{EQ} .

We have completed a 10-year longitudinal cohort study of noise and hearing damage that began in 2000.²³ We recruited first-year apprentices in the construction trades and a comparison group of unexposed university graduate students. We assumed apprentices would have limited prior exposure and good hearing at inception, allowing accurate

assessment of the noise-induced damage over the first years of noise exposure. Furthermore, the design included standard pure-tone behavioural threshold assessments and distortion product otoacoustic emissions (DPOAEs).

Findings at baseline indicated that the average construction apprentice had worked in construction for more than 2 years, had relatively high exposure to non-occupational and recreational noise and had elevated HTLs and depressed DPOAEs relative to controls.²³ After 5 years of follow-up, only limited evidence of additional noise-induced damage was observed. In comparison to controls, apprentices had slightly decreased emission amplitudes; however, the effect was partially due to apparent increases in emissions among the controls.²⁴ In order to provide additional exposure time and improve our ability to observe exposure-related changes, follow-up was continued for an additional 5 years. The extended follow-up also provided an opportunity to address several technical and logistical limitations identified in this first phase of the study, by making changes in the testing environment, equipment and task-based exposure assessment methods.

METHODS

Study design

The study was conducted in two phases spanning the years 2000–2010. At the beginning of phase 1, we recruited apprentices (carpenters, cement masons, electricians, ironworkers, insulation workers, masonry workers, operating engineers and sheet metal workers) at the start of their first year of training. Graduate students in the first year of their training at the University of Washington were recruited as non-noise exposed controls. Participants were given a baseline questionnaire, tympanometry, HTL and DPOAE tests. Tests were repeated approximately annually for 4 years.²³

Subjects who had completed at least two tests in the first phase were re-recruited to continue with annual tests for an additional 4 years. Phase 1 testing was conducted at the apprenticeship training sites in and around Seattle, while all phase 2 testing was conducted at the University of Washington, Research Center facilities. Results are described here for the full study period, and for phase 2 alone. Results for phase 1 were reported earlier.²⁴

Questionnaire

A questionnaire was administered at each annual visit, covering demographic characteristics, medical and otological histories and occupational and non-occupational noise exposure. Occupational noise exposure was evaluated with a task-based methodology for each job held as described below. Non-occupational noise exposure questions included the frequency of noisy activities and the use of hearing protection devices (HPDs) during these activities.²⁵

Tympanometry and audiometry

Subjects were screened with otoscopy and tympanometry to identify obstruction or middle ear problems that could affect the integrity of the audiometry. Participants with occluded canals were referred for cerumen management and asked to return at a later date.

Tympanograms were obtained over the pressure range of 200 to -400 daPa at 226 Hz. Participants with middle ear dysfunction (eg, flat tympanogram) were referred as indicated but continued with the study protocol. Subjects were counselled to use HPDs during any noise exposure prior to the test and asked if they had experienced any high exposure within the past 16 h.

In phase 1, audiometry was conducted by certified audiometric technicians (Washington Audiology Inc, Seattle, Washington, USA) in a test van meeting Occupational Safety and Health Administration (OSHA) requirements for background noise.²⁶ Audiometry was conducted using a Tremetrics RA 300 audiometer with TDH-39 headphones and an automated test sequence at 0.25, 0.5, 1, 2, 3, 4, 6 and 8 kHz. In phase 2, audiometric testing was conducted by research audiologists in a booth meeting American National Standard Institute (ANSI) criteria for audiometric test environments²⁷ at the University of Washington using a Grason-Stadler (Eden Prairie, Minnesota, USA) model GSI-61 audiometer with ER-3A insert earphones and calibrated to ANSI standards.²⁸ Audiograms were obtained at 0.5, 1, 2, 3, 4, 6 and 8 kHz following a standard bracketing procedure.

Distortion product otoacoustic emissions

In phase 1, DPOAEs were tested using the Bio-Logic (Knoxville, Tennessee, USA) Scout AuDX system with insert-mounted probes in a quiet room (<70 dBA) at the training sites. Emissions were measured at $2f_1$ - f_2 for target frequencies of f_2 =2, 3, 4, 6 and 8 kHz, and with f_2/f_1 set equal to 1.2. Response growth functions were collected for target L2 levels from 35 to 85 dB SPL in 5 dB steps, and L2 set equal to L1-10.

In phase 2, DPOAEs were tested in a booth using ER10C microphone insert probe assemblies with a custom measurement system.²⁹ The same level and frequency settings were used in phase 2; however, a different protocol was adopted. During phase 1, DPOAE measurements included clinically based stopping rules for test duration (from <1 to 16.4 s), and in phase 2, we adopted a uniform test period of 6 s for each level and frequency. Because the test environment, testing systems and assessment protocols were altered between phases, we conducted a calibration experiment for the two DPOAE systems.²⁹ Briefly, 78 ears were tested in random sequence with the two test systems with f_2 frequencies of 1, 2, 3, 4, 6, and 8 kHz and with L2 from 25 to 85 dB. Differences between the two generated growth functions were minimised by calibration of the emission amplitude (vertical transformation) or the stimulus levels (horizontal transformation). Horizontal transformation produced the best fit of the curves and resulted in correlations (r) of 0.81 (8 kHz) to 0.89 (2 kHz) between phase 1 and phase 2 measurement system results. Using this frequency-specific calibration, DPOAE amplitudes from phase 1 were adjusted to conform with the phase 2 measurement data.

Exposure assessment

A detailed description of the development of task-based exposure estimates for this cohort is reported elsewhere.³⁰ Briefly, for each subject, the amount of time reported in each task was summed across all jobs within each subject-interval (eg, the time between questionnaires).

Information on task durations within a subject-interval was combined with task-specific noise levels using equation 1:

$$L_{EQij, TB2000} = 10 \log_{10} \left[\frac{1}{2000 \times Y_{ij}} \left(\left(\sum_{t=1}^T H_{ijt} 10^{L_t/10} \right) + \left(H_{NCij} \times 10^{L_{NC}/10} \right) \right) \right], \quad (1)$$

where L_t is the mean L_{EQ} level for task t and is applied to H hours for which that task was reported by individual i in subject-interval j within a subject-interval of Y years in duration. The second term in the equation relates to reported non-construction (NC) hours in noisy jobs, which were assigned an arbitrary L_{EQ} noise level L_{NC} of 85 dBA. The task-specific noise levels were developed from 1310 full-shift noise measurements collected between 1997 and 2008 on commercial construction sites in the Seattle area. Several noise metrics were estimated for each task, and L_{EQ} was adopted for the primary exposure metric and L_{max}/L_{EQ} ratio was used to test the additional effect of exposure peakiness.⁴ Subject-interval estimates were normalised to an annual 2000-h exposure period to account for the large variability in hours worked in each interval and in interval lengths.³⁰

Non-occupational noise exposure was estimated following the methods presented earlier.²⁵ The interval-specific duration of non-occupational activities were combined with average estimated exposure levels to obtain a crude estimate of an annual 2000-h equivalent noise exposure. This distribution was then classified into low (<80 dBA), medium (80–90 dBA) and high (>90 dBA) for analysis.

Statistical analysis

Analysis was conducted on ears with two sets of tests with hearing thresholds <50 dB HL (mean 0.5, 1 and 2 kHz) and no significant middle ear problems. An audiologist (PF) reviewed 48 audiometric files with >20 dB change in HTL within one time interval and excluded aberrant tests, with identifiable reasons such as injuries, illnesses or test problems.

We selected audiometric thresholds and DPOAEs at 3, 4 and 6 kHz (DPOAEs with L2=40 dB) as the primary outcomes because of their presumed sensitivity to noise-damage. Descriptive analyses compared subjects who were construction apprentice students as ‘exposed’ and ‘unexposed’ groups, respectively, and change over time was assessed among the subset with six or more tests. Controlled analyses addressed all subjects and used 2000-h adjusted L_{EQ} noise levels, estimated on an interval-specific individual basis and averaged over time since baseline.

Linear mixed models were developed to estimate the change in hearing level in each ear over time in relation to noise, while controlling for subject-specific baseline covariates, age (<30 years, 30 years), gender and hearing threshold (average HTL at 3, 4 and 6 kHz; <10, 10–20, >20 dB). All models included random intercepts for subject and dominant ear nested within subject (thus controlling non-independence of a subject’s ears) and a random slope for years since baseline at the subject level. Models were run for phase 2 data only, and for phases 1 and 2 combined including a dummy variable for phase. The model form is:

$$Y_{it,l} = \alpha_0 + (b_{0i} + b_{0i,l}) + (\beta_1 + b_{1i}) T_{it} + \beta_2 X_{it} T_{it} + \gamma Z_{it,l} + \varepsilon_{it,l}, \quad (2)$$

where i indexes subject, l indexes ear (dominant or non-dominant hand side) and t indexes the visit time since baseline. The term T_{it} indexes time in years since baseline, X_{it} is the subject's exposure (average L_{EQ} since baseline -70 dBA) and $Z_{it,l}$ are the baseline and phase fixed-effects adjustment variables. Seventy dBA was chosen as the reference value because no NIHL would be expected below this level regardless of exposure duration.³¹ The b 's represent the random effects and ε the error term. Models were developed for each of two outcomes (HTL at 4 kHz, DPOAE at 4 kHz and 40 dB), for phases 1 and 2 data combined and for phase 2 alone.

There were two time-related terms in these models: years since baseline and the interaction of noise exposure and years since baseline (in dB×years), representing cumulative exposure. By including these two variables, we simultaneously estimated the change in hearing outcome over time (β_1 , ie, ageing alone) and the additional effect of noise exposure on the change in hearing over time (β_2). Our primary measure of the effect of noise on hearing is estimated by an increase of 10 dB of exposure over a 10-year period (ie, $\beta_2 \times 10 \times 10$). Selected models were rerun for the additional outcomes (HTL: 3, 4 and 6 kHz, and DPOAEs at f2=3, 4 and 6 kHz and L2=40), for both phase 2, and phases 1 and 2 combined.

The peakiness exposure metric was classified into a binary variable to indicate high peakiness over the study period (L_{\max}/L_{EQ} vs >50). High peakiness was added to our base models at 4 kHz as an interaction with our primary exposure variable, thus demonstrating the effect of an increase in exposure (L_{EQ}) within the subgroup defined by high levels of peakiness.

Several sensitivity analyses were conducted: models were run on exposed subjects only because of differences in exposure history and hearing at baseline compared with control subjects and to address any uncontrolled confounding by socioeconomic status; 4 kHz models were re-run excluding tests at which the subject reported 'high' noise exposure within the last 16 h; 4 kHz models were also rerun after stratifying on HTL at baseline, and by average noise level (<85 dBA, 85 dBA); and base models were rerun excluding subjects with low DPOAEs at baseline, as defined by an emission amplitude <6 dB above the noise floor.

RESULTS

Of the 456 subjects tested at baseline, 342 returned for at least one additional test. Exclusion criteria eliminated ears with peak pressure less than -100 or >100 daPa ($n=204$ tests), large low frequency (0.5, 1 and 2 kHz) hearing losses > 50 dB HL ($n=11$ tests) or ear canal volumes >3.0 cm³ (as measured at 226 Hz; $n=28$ tests). Review of the audiometric files revealed an additional 12 tests with large changes such as injury and test errors; these tests were also removed. After dropping ears with only one remaining test, there were 316 individuals (617 ears) and 2896 total ear-specific test events (an average of 4.7 ± 2.0 tests per ear), 1008 of which were during phase 2 of the study.

We compared the 316 subjects in analysis with the 140 excluded for baseline characteristics. Excluded subjects were slightly younger (26.5 ± 7.7 vs 27.6 ± 3.3 years, $p=0.09$), had non-occupational exposure more frequently (75% vs 66%, $p=0.06$) and had slightly worse hearing (average 3, 4, 6 kHz HTL: 14.4 ± 14.4 vs 13.0 ± 11.9 , $p=0.13$; 4 kHz DPOAE: -22.7 ± 8.2 vs -21.8 ± 8.5 , $p=0.12$) but were otherwise very similar.

Exposed workers were similar to the non-exposed in terms of age, and duration of follow-up, however, they differed significantly in gender, and prior exposure (table 1). At baseline, construction workers had an average HTL more than 7 dB higher than controls at 4 kHz and lower DPOAE amplitudes. Noise exposures for workers were high (87.2 ± 3.6 dBA), while controls were assigned an annualised base exposure of 70 dBA reflecting their office work environments.

HTLs and DPOAEs over the duration of the study are displayed in figure 1. Note the 1.5-year gap in data collection between the two phases. Changes in exposed and unexposed groups are close to parallel, indicating a relatively modest average effect of noise exposure. There is an unexpected decrease in HTL at 6 kHz, suggesting a mild improvement in hearing, although an analogous change in DPOAE at 6 kHz was not observed.

Among ears with six tests, changes in HTLs and DPOAEs ($L2=40$ dB) are shown for the first and last tests (figure 2). Mean, 2.5th and 97.5th percentiles are shown across frequencies for 182 worker's ears and 38 control's ears. The change in mean HTL is greater among workers at frequencies below about 6 kHz, and for DPOAEs above about 3 kHz.

In linear mixed models for 4 kHz outcomes, non-occupational noise exposure at baseline or during the study period, and years of construction work at baseline had no effect on the results and were dropped from analysis. Subjects with higher age and higher hearing thresholds at baseline had higher 4 kHz thresholds (table 2). In the phases 1 and 2 combined model hearing thresholds increased over time, but not significantly, (0.025 ± 0.14 dB per year), however, in the phase 2 model, thresholds increased significantly (0.65 ± 0.53 dB per year). The effect of noise exposure was small but significant, $0.023 (\pm 0.008)$ and $0.034 (\pm 0.013)$ dB HTL/dB \times year, in the phases 1 and 2, and phase 2 models, respectively, with consistent effects of noise exposure across models.

Modelled results for 4 kHz DPOAE amplitude also showed expected differences with age and increasing HTLs at baseline (table 2). Similar patterns are observed for DPOAEs, with the time effect small in the phases 1 and 2 model, and more pronounced in the phase 2 model. The noise effect for the two models is very consistent (-0.01 ± 0.006 and -0.02 ± 0.008 dB per dB \times year), though lower than the effect observed for HTL.

For the four models, the SD for the random intercept and exposure slope for subject, and the random intercept for ear within subject, as well as the correlation between the subject intercept and exposure slope are given in table 2. The random effects and their uncertainties are larger for the phase 2 models, demonstrating the combined effects of data collected more distant in time from baseline, smaller sample size and data collected over a shorter time. Random variation in intercepts between subjects is high (approximately 5–10 dB), and there is considerable variation between ears nested within subject (2.6–7.2 dB). Similarly, the

interindividual variation in slope is large (0.45–1.32 dB) compared to the SD of the ageing effect. The residual variation SD was about 4 dB for HTL and 6 dB for DPOAEs.

The estimated effects of an increase of 10 dB in average exposure level over 10 years at 3, 4 and 6 kHz are presented in table 3. The noise effect during phase 2 compared with phases 1 and 2 is very consistent, especially given the differences in the effect of time. The effect of noise on HTL at 4 and 6 kHz was somewhat higher in phase 2 and almost the same at 3 kHz for both HTL and DPOAE. The overall effect on HTL is approximately 2.5–3 dB of hearing loss for an additional 10 dB exposure over 10 years. A somewhat smaller effect of noise on DPOAEs was observed, on the order 1–2 dB decrease per 10 years for a 10 dB exposure increase.

The 4 kHz models incorporating an interaction term between noise exposure and an indicator variable for high ‘peakiness’ estimate the effect of noise on hearing over time for those with low (n=168 subjects) and high (n=148 subjects) levels of exposure peaks (table 3). For the HTL outcome, there is evidence that exposure with a high peakiness component has a greater effect (2.4 vs 1.2 dB); however, this effect is not evident in the phase 2 analysis. The opposite pattern is observed for DPOAE—with an additional effect of peaky noise observed only for phase 2. Thus, there are inconsistent results for the effect of high peak component noise on hearing.

Compared with models with all subjects, the effect of exposure among construction workers only on DPOAE was reduced from $-1.96 (\pm 0.880)$ to $-0.257 (\pm 1.63)$ in the phase 2 only model. Other results were negligibly affected (data not shown). Models were run excluding the 253 tests for which the subject reported having substantial noise exposure within the prior 16 h (whether they wore HPDs). The exposure effect estimates were either unchanged or demonstrated slightly higher effects with a concomitant increase in statistical significance. In models excluding 169 ears with baseline 4 kHz DPOAEs <6 dB above the noise floor, no substantial change was observed in the estimated effect of exposure. No significant difference in noise effect was observed for subjects with average exposures less than versus greater than 85 dBA. We also re-ran models with several alternative methods of adjusted for self-reported HPD use (including a subanalysis on the subgroup who reported never using HPDs) and found negligible changes in effect measures, thus confirming our observation of poor compliance and inaccurate reporting of HPD use.

Interestingly, when 4 kHz models were rerun stratifying on baseline hearing level (HTL average of 3, 4 and 6 kHz; <10, 10–20 and 30 dB), a trend of increasing effect of noise with increasing HTL at baseline was observed. For the combined phases 1 and 2 model, estimated noise-related hearing loss was 1.4 (± 0.73), 2.62 (± 1.64) and 6.89 (± 3.24) dB per 10 dB exposure over 10 years for those with low, medium and high HTLs at baseline, respectively. No comparable trend was observed for DPOAEs.

DISCUSSION

This study is among the only studies designed to assess the effects of noise on hearing, beginning with a relatively naive cohort and following subjects for up to eight annual tests

over a 10-year period. The cohort included construction workers who have relatively high noise exposure levels, including a high degree of peak noise, but who wear HPDs a small fraction of the time.³² The analysis is based on an exposure measurement database of over 1300 full-shift personal noise dosimetry measurements on subjects and construction workers doing comparable work.³⁰ However, the long duration of the study and necessary changes to study protocols and testing equipment make the longitudinal analysis challenging. We addressed these issues by calibration of the test systems to the degree possible, but acknowledging that these procedures could not perfectly control differences, we added a dummy variable to the models to address the two phases of data collection. To the degree that these challenges were overcome, we observed an average of 2–3 dB worsening in HTLs at 4 kHz for a 10 dB increase in exposure over 10 years, after accounting for previous hearing loss, age and gender differences. Similarly, we observed about 1–2 dB decrease in DPOAE amplitude at 4 kHz for a 10 dB increase in exposure over 10 years.

Findings are quite robust in our analysis; however, the relatively small noise-related change in hearing over 10 years was somewhat surprising. Several factors contributed to this; subjects were not ‘naive’ but had had previous work and recreational exposures, and a hearing loss of about 12 dB at 4 kHz.²³ Given this starting point, the standard ANSI model for NIHL predicts 1 dB additional NIHL after an additional 10 years of exposure at 87 dBA.²⁰ Thus, the amount of noise-related loss observed is larger than the model would predict. We also saw evidence that those with higher hearing thresholds at baseline suffered a larger noise-related decline, raising the possibility that subgroups have increased susceptibility to NIHL over extended periods of exposure. Genetically based susceptibility to noise has been described in both mouse models and human populations (see Konings *et al* 2009³³). It is also possible that individuals with poorer hearing at baseline were less consistent users of HPDs, though given the low prevalence of HPD use in the study group, and lack of evidence of effect of HPD use, we do not believe protective devices could be responsible for this large difference in effect.

Our prior hypothesis, that noise exposure with a high peak component would produce greater hearing damage than relatively constant noise was minimally supported. HTLs increased at a greater rate among those with high peak exposures, but the effect was not observed when restricted to phase 2, and the opposite pattern was seen for change in DPOAE. Other evidence for the disproportional impact of high impact noise signals comes from cross-sectional studies comparing workers in processes with high and low kurtosis noise signals.³⁴ Unfortunately, our study cannot provide definitive resolution to this significant concern about damaging noise signals.

A strength of this study was the exposure quantification using over 1300 full-shift measurements. We adopted a task-based approach to individual estimation having demonstrated its relative accuracy in validation studies.³⁵ These estimates combined the 179 task-specific exposure levels with individually reported task times, resulting in individual-specific exposures. Still, the noise exposures are only estimates, and thus, our exposure effect estimates may still be influenced by measurement error.³⁶

Because the self-reported HPD use is so unreliable, and the actual use is relatively low, we have chosen to not adjust for HPD use in our main analyses. However, we did attempt several methods of accounting for HPDs, including restricting the analysis to those that reported never using HPDs, which is most likely to be accurate, and found no significant change in our effect estimates. In our previous analyses of HPD use among construction workers, we have found 50% of construction workers self-reported ‘always’ use, but when observed, the fraction of exposure time in which HPDs were used was only about 17%³⁷ to 24%.³²

This study produced no evidence that DPOAEs could be used to detect noise-induced damage more sensitively or at an earlier stage than standard behavioural hearing thresholds. The overall effect of noise on DPOAEs was small but appeared earlier in the study (phase 1) than did elevations in pure-tone thresholds.²⁴ Furthermore, DPOAEs were less robust in the apprentices than in controls, raising the possibility that any early detection advantage offered by the DPOAEs may be undermined by changes that had occurred prior to the onset of the study. This leaves open the possibility that DPOAEs may be effective in identifying damage at young ages or before substantial damage is accrued.

The models we adopted for analysis estimated the average change in hearing per decibel of increased exposure—thus producing a linear effect estimate, which is contrary to our understanding of NIHL. For instance, our model would estimate the same effect for each decibel increase in exposure for an individual at 75 or 95 dBA. Initial exploration of the expected non-linearity of effect expected over this range did not produce clear results, suggesting that our study is inadequately powered to detect the non-linear effects, particularly in the presence of exposure measurement error.

CONCLUSIONS

Despite the challenges encountered, the results of this longitudinal study demonstrated noise-induced damage to hearing among construction workers exposed to levels close to the current exposure limit of 85 dBA. The effect was consistent with or exceeded that predicted by the prevailing model for NIHL. The effect was modest, on the order of an estimated 2–3 dB over a 10-year period for an increase in 10 dBA, most likely due to the significant excess hearing loss present among these workers at baseline. Although these subjects continued to lose hearing acuity at an increased rate over the study period, they had already suffered a large fraction of their overall loss prior to entering into the study. Our results reinforce the need for enhanced hearing loss prevention programmes and noise control efforts in the construction industry, and the need to begin hearing prevention activities as early as possible in the lives of young workers.

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What this study adds

- A large group of construction apprentices were observed over a 10-year period with both standard audiometry and distortion product otoacoustic emissions, while characterising their noise exposure.
- A greater than expected effect of noise exposure on changes in hearing thresholds was observed among this group, on the order of 2–3 dB for an increase in 10 dBA of exposure over 10 years.
- DPOAEs did not provide additional sensitivity to the ability to assess damage over time.
- Construction workers exposed to noise near the current exposure guidelines continue to suffer noise-induced hearing damage. The greatest extent of hearing loss is suffered early in life, presumably because of occupational and non-occupational noise exposure at an early age.

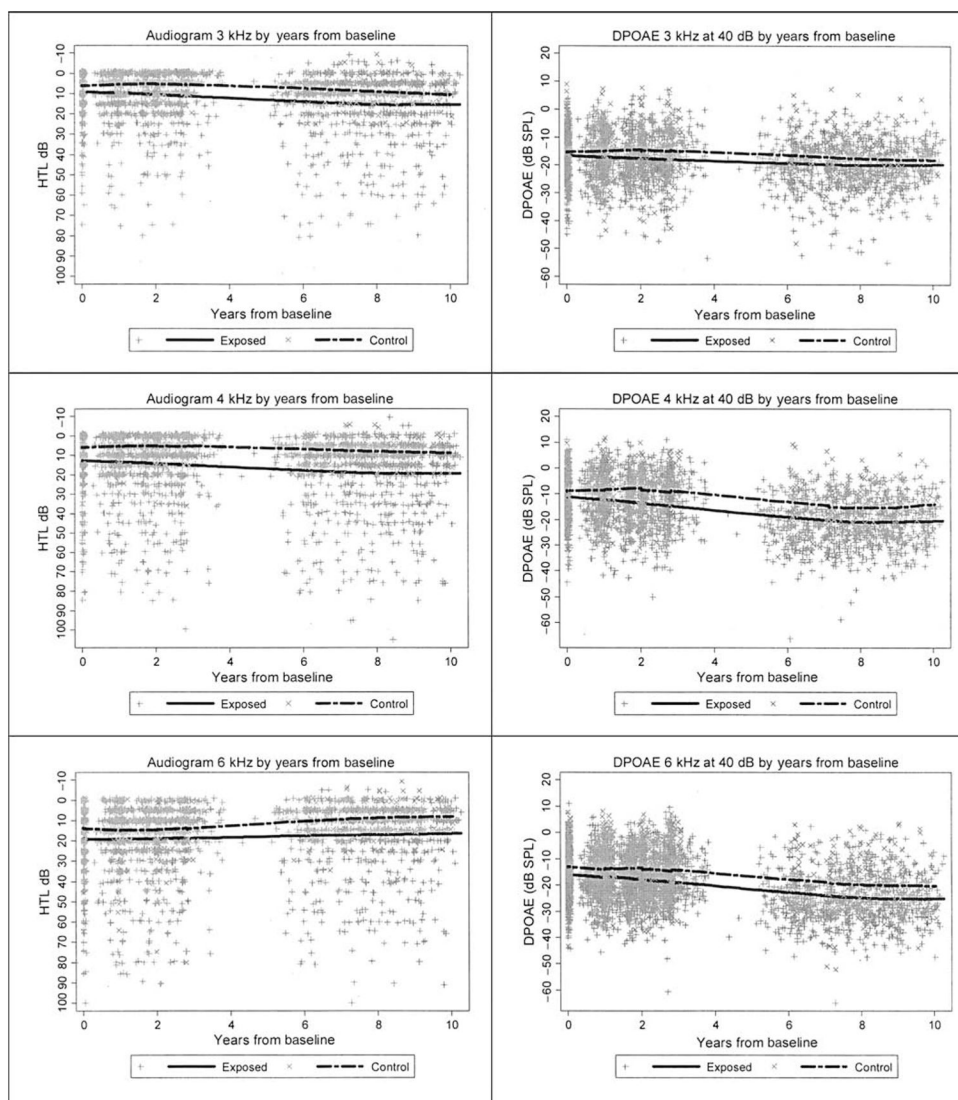


Figure 1. Hearing threshold levels (HTL) and DPOAEs (40 dB) over time.

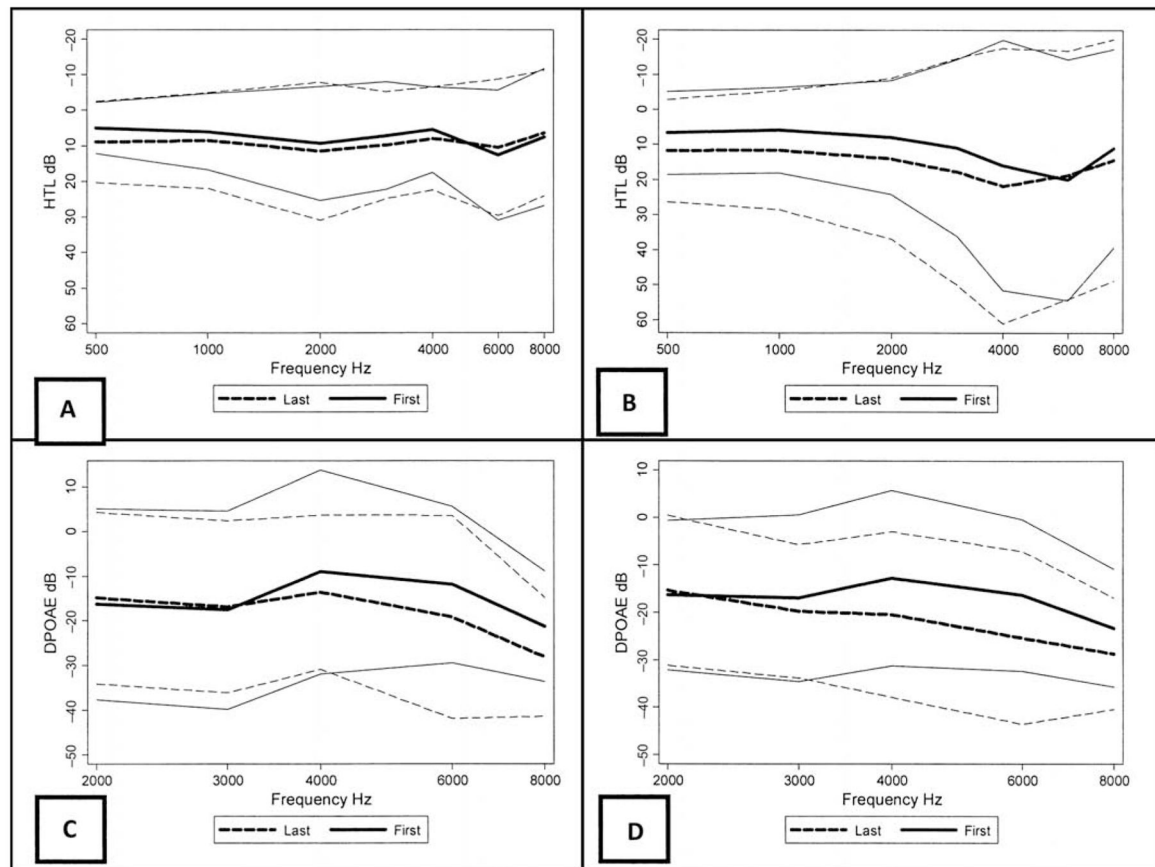


Figure 2.

Average and 95% CI hearing threshold levels (HTLs) and DPOAEs (L2=40 dB) at baseline and last test among subjects with six or more tests. (A) HTLs, controls (n=38 ears), (B) HTLs, construction workers (n=182 ears), (C) DPOAEs, controls (n=38 ears), (D) DPOAEs, construction workers (n=182 ears).

Table 1

Characteristics of study subjects

	Construction workers	Control subjects	All
Demographics, N subjects	258	58	316
Age	27.6 (6.6)	27.4 (4.3)	27.6 (3.3)
Gender (male)	229 (89%)	31 (53%)	261 (83%)
Prior construction work (years)	2.5 (3.4)	0.1 (0.6)	2.1 (3.2)
Prior non-occupational noise	187 (72%)	22 (38%)	209 (66%)
Exposure			
Duration of follow-up (years)	5.6 (3.5)	5.1 (5.5)	5.5 (3.5)
Average annualised L_{EQ}	87.2 (3.6)	70.0 (0)	84.1 (7.4)
Exposure peakiness (L_{max}/L_{EQ})	49.9 (19.8)	0	40.8 (26.35)
Baseline hearing threshold, N ears	502	115	617
3 kHz	9.3 (10.8)	6.2 (6.2)	8.7 (10.2)
4 kHz	13.1 (15.7)	5.8 (6.8)	11.7 (14.7)
6 kHz	19.3 (15.9)	13.8 (10.7)	18.2 (15.2)
DPOAE, 40 dB			
3 kHz	-16.5 (8.3)	-15.4 (9.3)	-16.3 (8.5)
4 kHz	-11.2 (9.2)	-8.8 (9.5)	-10.8 (9.3)
6 kHz	-16.2 (8.4)	-12.7 (9.2)	-15.6 (8.7)

Table 2

Linear mixed models for 4 kHz HTL and DPOAE*

4 kHz HTL	Phases 1 and 2		Phase 2 only	
	Estimate (SE)	p Value	Estimate (SE)	p Value
Fixed effects				
Phase 2	2.22 (0.550)	<0.001	–	
Age (>30 years)	3.03 (0.901)	0.001	6.90 (1.66)	<0.001
Gender (male)	2.05 (1.05)	0.050	0.883 (2.10)	>0.2
HTL at baseline (ref <10)				
10–20	7.50 (0.788)	<0.001	6.63 (1.37)	<0.001
>20	30.2 (1.09)	<0.001	31.0 (1.91)	<0.001
Years since BL (years)	0.025 (0.139)	>0.2	0.648 (0.53)	0.010
Noise exposure × years (dB×years)	0.023 (0.008)	0.003	0.034 (0.013)	0.011
Random effects				
Subject: random intercept SD	4.78 (0.481)		9.57 (1.45)	
Subject: random slope SD	0.735 (0.54)		1.32 (0.174)	
Subject: intercept–slope corr.	0.024 (0.114)		–0.688 (0.092)	
Ear: random intercept SD	6.46 (0.302)		7.21 (0.472)	
Residual SD	4.20 (0.065)		3.75 (0.112)	

4 kHz DPOAE (40 dB)	Phases 1 and 2		Phase 2 only	
	Estimate (SE)	p Value	Estimate (SE)	p Value
Fixed effects				
Phase 2	–6.64 (0.762)	<0.001	–	
Age (>30 years)	–2.45 (0.737)	<0.001	–2.56 (1.12)	0.023
Gender (male)	–3.61 (0.875)	<0.001	–3.24 (1.46)	0.026
HTL at BL (ref <10)				
10–20	–3.83 (0.531)	<0.001	–3.13 (0.993)	<0.002
>20	–8.79 (0.778)	<0.001	–5.00 (1.34)	<0.001
Years since BL (years)	0.059 (0.143)	>0.2	0.516 (0.221)	0.020
Noise exposure × years (dB×years)	–0.011 (0.006)	0.083	–0.020 (0.008)	0.026
Random effects				
Subject: random intercept SD	5.23 (0.338)		9.42 (2.31)	
Subject: random slope SD	0.450 (0.063)		0.762 (0.369)	
Subject: intercept–slope corr.	–0.375 (0.119)		–0.914 (0.067)	
Ear: random intercept SD	2.60 (0.262)		4.75 (0.437)	
Residual SD	6.13 (0.095)		5.68 (0.167)	

* Effect estimates in dB per unit change of each predictor.

Table 3Estimated effect in dB on hearing over 10 years, and 10 dB (L_{EQ}) increase in exposure*

Variable	HTL		DPOAE at 40 dB	
	Phases 1 and 2	Phase 2 only	Phases 1 and 2	Phase 2 only
3 kHz HTL (effect (SE))	2.24 (0.746)	2.16 (1.16)	-0.92 (0.611)	-0.408 (0.812)
p Value	0.003	0.062	0.133	>0.2
95% CI	0.777 to 3.70	-0.105 to 4.42	-2.12 to 0.279	-2.00 to 1.19
4 kHz HTL (effect (SE))	2.33 (0.77)	3.38 (1.32)	-1.11 (0.64)	-1.96 (0.880)
p Value	0.003	0.011	0.083	0.026
95% CI	0.813 to 3.85	0.784 to 5.97	-2.36 to 0.045	-3.68 to -0.232
6 kHz HTL (effect (SE))	1.60 (0.958)	2.91 (1.36)	-1.30 (0.721)	-1.01 (0.985)
p Value	0.094	0.033	0.072	>0.2
95% CI	-0.275 to 3.48	0.242 to 5.57	-2.71 to 0.116	-2.94 to 0.921
4 kHz (effect (SE))				
Exposure with low peakiness [†]	1.17 (0.885)	3.64 (1.47)	-1.43 (0.787)	-1.03 (1.02)
95% CI	-0.565 to 2.91	0.766 to 6.52	-2.98 to 0.105	-3.03 to 0.956
Exposure with high peakiness [†]	2.40 (0.772)	3.38 (1.33)	-1.07 (0.646)	-2.13 (0.873)
95% CI	0.886 to 3.91	0.774 to 5.98	-2.34 to 0.194	-3.84 to -0.41

* Models controlled for time since baseline, age, gender and baseline hearing threshold, and phase, where appropriate.

[†] Additional effect of noise exposure with high peakiness, interaction term.